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CASE REPORT

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Secondary aortoenteric fistula possibly associated with continuous physical stimulation: a case report and review of the literature

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Abstract

Background: Secondary aortoenteric fistula is a rare but fatal complication after reconstructive surgery for an aortic aneurysm characterized by abdominal pain, fever, hematochezia, and hematemesis, and the mortality rate is high. It has been suggested that it arises due to either continuous physical stimulation or prosthesis infection during primary surgery. We describe an aortoenteric fistula following reconstructive surgery for an abdominal aortic aneurysm together with postmortem pathological findings.

Case presentation: A 59-year-old Japanese man who had undergone reconstructive surgery for an abdominal aortic aneurysm 20 months earlier presented with the chief complaint of hematochezia and malaise. Esophagogastroduodenoscopy and total colonoscopy revealed only colon diverticula with no bleeding. Contrast-enhanced computed tomography revealed gas within the aneurysm sac and adhesion between the replaced aortic graft and intestinal tract, suggesting a graft infection. After 18 days of antibiotic treatment, he suddenly went into a state of shock, with massive fresh bloody stool and hematemesis, followed by cardiac arrest. An autopsy revealed communication between the artery and the ileum through an ulcerative fistula at the suture line between the left aortic graft branch and the left common iliac artery. Pathological analysis revealed tight adherence between the arterial and intestinal walls, but no marked sign of infection around the fistula, suggesting that the fistula had arisen due to physical stimuli.

Conclusions: Pathological analysis suggested that the present secondary aortoenteric fistula arose due to physical stimuli. This reaffirms the importance of keeping reconstructed aortas isolated from the intestine after abdominal aortic aneurysm surgery.

Keywords: Aortoenteric fistula, Abdominal aortic aneurysm, Intestinal bleeding, Herald bleeding

Background

A secondary aortoenteric fistula (SAEF) is an abnormal connection between the aorta and gastrointestinal tract resulting from reconstructive surgery for an abdominal aortic aneurysm (AAA), including open repair surgery and endovascular treatment. Although rare (incidence rate, 1.6 to 4%) [1–4], it is life-threatening and has a

high mortality rate (24 to 45.8%) [5–7]. Elucidating the clinical features and improving the diagnosis of SAEF would therefore be valuable.

The mechanism underlying the pathological development of SAEFs remains unknown. Two hypotheses have been postulated. One suggests an origin in continuous stimulation due to aortic pulsation directly affecting the walls of the intestinal tract and arteries [2, 4, 7–9]. This theory is supported by the fact that most SAEFs involve the third or fourth portion of the duodenum which are compressed between the superior mesenteric artery and

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abdominal aorta in the retroperitoneal space [5]. The other argues an origin in a local inflammatory response due to prosthesis infection during primary surgery [5, 7–10]. Several species of bacteria not usually identified in the intestine, such as *Staphylococcus*, have been detected in aortic prostheses in cases of SAEF, which supports this hypothesis.

Here, we describe an aortoenteric fistula following reconstructive surgery for an AAA and discuss the implications of postmortem pathological analysis. We believe the findings further our understanding of the underlying mechanism of SAEFs, which could be useful in deciding a preventive strategy.

Written informed consent was obtained from the patient's family for publication of this case.

Case presentation

A 59-year-old Japanese man presented to our hospital with the chief complaint of hematochezia and malaise. On the day of admission and 10 days earlier, he had produced a fresh bloody stool. He had undergone open surgery with a bifurcated graft for an AAA 20 months earlier. The course was uneventful, with no remarkable findings on computed tomography (CT) at 6 and 18 months postoperatively. An abdominal examination at our hospital revealed nothing remarkable and no tenderness. His blood pressure was 122/75 mmHg; heart rate, 86/minute; body temperature, 36.6 °C; breathing, 16 per minute; and hemoglobin level, 9.0 g/dL.

Esophagogastroduodenoscopy and total colonoscopy revealed only colon diverticula and no bleeding. Contrast-enhanced CT revealed gas within the aneurysm sac (Fig. 1a, b, yellow arrowhead) and adhesion between the graft and intestinal tract in three areas: the ileum had attached to the anastomosis between the left branch of the graft and left common iliac artery (Fig. 1a, yellow circle); the jejunum to the middle of the graft body; and the duodenum to the anastomosis between the aorta and the proximal graft. *Enterococcus faecium* was isolated from blood culture, suggesting communication

between the intestinal tract and aorta at the attached sites, possibly due to infection of the graft. His vital signs were stable, so surgery was scheduled to take place after antibiotic treatment. After admission, he produced another fresh bloody stool, but bleeding ceased immediately. At 18 days after the second fresh bloody stool, however, he suddenly went into a state of shock, with massive fresh bloody stool and hematemesis, followed by cardiac arrest. Despite intensive cardiopulmonary resuscitation, he died from hemorrhagic shock.

An autopsy performed with written consent from the family revealed an ulcerative fistula in the distal ileum that adhered to the anastomosis between the left branch of the graft and the left common iliac artery (Fig. 2a), with a small hole at the aortic anastomosis (Fig. 2b). Arterial structure was destroyed at the anastomotic site. There was fibrous thickening of the arterial wall (Fig. 3a, b) and the external elastic lamina had disappeared. The serosa of the small intestine and adventitia of the artery were firmly adhered (Fig. 3c, d). There was no marked sign of infection, such as inflammatory cells or phagocytosis, even around the fistula.

Discussion

SAEFs are a rare but life-threatening complication after aortic aneurysm reconstruction [1–3, 11]. Despite the importance of establishing the underlying mechanism of fistula formation, their etiology remains unclear. It has been suggested that either chronic infection of the graft or physical stimulation, such as from aortic pulsation pressure, contributes to their formation, which is usually determined based solely on the clinical course or surgical findings [12–19]. To the best of our knowledge, only 25 case reports on SAEFs have been published (see Table 1). Of these, only six discuss the possible mechanism of fistula formation and the results of pathological examination [20–25]; and only two of these reports include detailed pathological images [20, 25]. Therefore, the precise mechanism underlying fistula formation remains to be clarified pathologically.

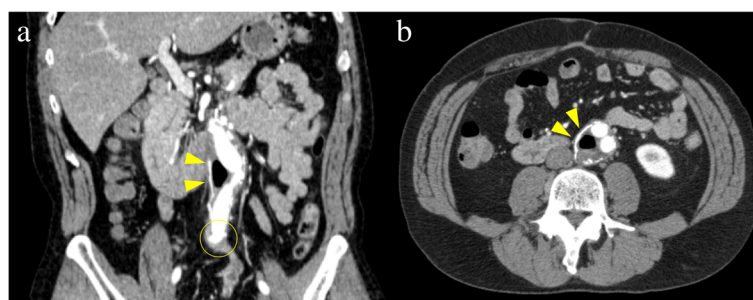


Fig. 1 Contrast-enhanced computer tomography (coronary (a) and axial (b) view) revealed gas within aneurysm sac (yellow arrowhead), and attachment of ileum to anastomosis between left branch of graft and left common iliac artery (yellow circle)

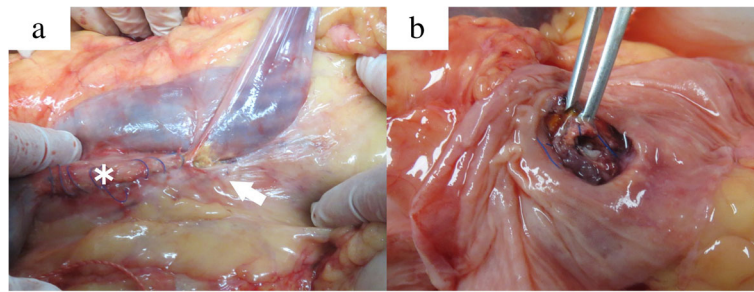


Fig. 2 Image at autopsy. Adhesion of the distal ileum to the anastomosis between the left branch of the graft and the left common iliac artery (arrow), and the postoperative aneurysm sac on the head side (asterisk) (a), with a small hole at the aortic anastomosis in dissected ileum (b)

The present autopsy findings suggested that physical stimuli following postoperative adhesion and attachment between the iliac artery and small bowel had contributed to the formation of the fistula, although chronic infection of the graft could not be excluded completely. Despite fibrillar change in the serous membrane and muscularis propria layer of the intestine, inflammatory cell infiltration was sparse at the edge of the fistula. Moreover, there was no sign of phagocytosis, indicating no destructive infection of the graft. The arterial wall exhibited fibrous thickening, however, possibly due to continuous physical stimulation. These pathological findings and the clinical course suggested that the persistent pulsation of the aorta had physically degraded the attached intestinal wall and aortic membrane, inducing erosion of the bowel tract. Fatal bleeding appears to have occurred when the vulnerable inner membrane of the attached aorta finally ruptured.

In terms of the underlying mechanism of fistula formation, the present findings reaffirm the importance of avoiding adhesion between the intestinal wall and the anastomosis of the graft due to insufficient isolation of the latter [26]. Interposing native tissue between the aorta and the intestinal tract at primary surgery has been reported as effective in preventing SAEF associated with physical stimuli [1]. In the present case, the artery and intestinal tract had adhered tightly at the site of the fistula, even though the artificial graft had been covered with a longitudinally split aneurysmal wall and retroperitoneum during primary surgery. This particular fistula involved both the ileum and the distal suture line of the graft, a relatively rare site for an SAEF. The ileum might have become trapped by adhering to the closing line of the retroperitoneum after primary surgery, resulting in tight contact with the distal anastomosis of the graft. This suggests that keeping a graft isolated from the

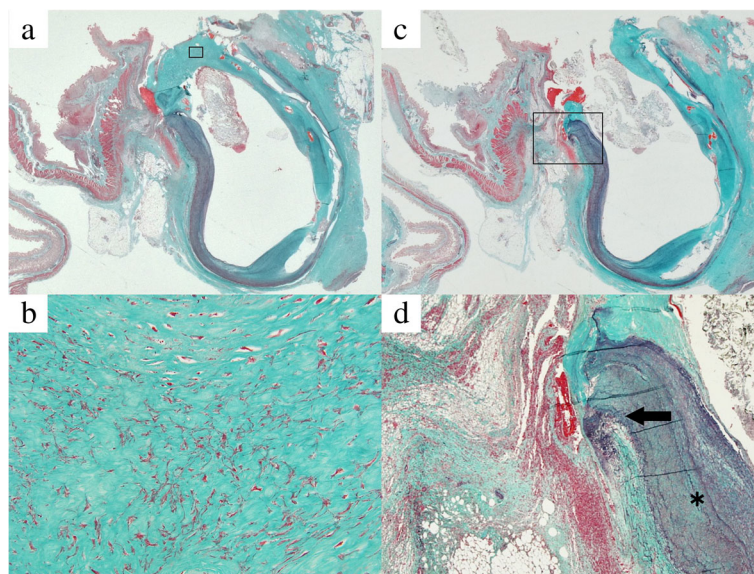


Fig. 3 Histology of left common iliac artery by Elastica van Gieson stain revealed fibrotic and thickened intima of anastomosed artery exposed to intestinal lumen at edge of fistula (a whole, magnification $\times 1$; b square, magnification $\times 400$). Histology of fistula revealed firm adhesion (arrow) between serosa of intestinal tract and adventitia of artery (asterisk) (c whole, magnification $\times 1$; d square, magnification $\times 40$)

Table 1 Summary of published case reports describing secondary aortoenteric fistula after both open surgery and endovascular aortic reconstruction for abdominal aortic aneurysm

Date	Authors and reference number	Age, sex	Clinical presentation	Initial treatment for aneurysm	Period [†]	Treatment for SAFE	Outcome	Pathological examination [‡]	Estimated aetiology [§]
1	2018 Anwon et al. [20]	42 M	Hematemesis, melena	EVAR	1 yr 1 m	Aortic reconstruction, bowel repair	Alive at 9 months	+	Mechanical factor
2	2018 Alfawaz et al. [28]	62 M	Malaise, fever	Open surgery	7 yr	Aortic reconstruction, bowel repair	Alive at 7 days	–	Unclear
3	2017 Dickfos et al. [19]	75 M	Abdominal pain, fevers	EVAR	1 yr 2 m	Aortic reconstruction	Unclear	–	Infection
4	2016 Kadhim et al. [21]	66 M	Confusion, fever	EVAR	15 yr	Bowel repair, debridement	Alive at 12 months	+	Infection
5	2017 Guevara-Noriega et al. [18]	65 M	Malaise	Open surgery	1 yr 10 m	Aortic reconstruction, bowel repair	Alive at 2 years	–	Infection
6	2014 Zaki et al. [17]	75 M	Abdominal pain, hematemesis	EVAR	2 yr 6 m	Aortic reconstruction, bowel repair	Died	–	Infection
7	2013 Jamal et al. [29]	60 F	Hematemesis, melena	Open surgery	6 yr	Surgery	Died	–	Unclear
8	2009 Grassia et al. [30]	72 M	Hematochezia	Open surgery	8 yr	Aortic reconstruction	Alive at 2 weeks	–	Unclear
9	2009 McAloon et al. [31]	73 M	Lethargy, melena	Open surgery	10 yr	None*	Died	–	Unclear
10	2008 Hara et al. [16]	84 M	Hematochezia	Open surgery	14 yr	Aortic reconstruction, bowel repair	Alive at 6 months	–	Recurrent aneurysm
11	2008 Geraci et al. [15]	59 M	Dyspepsia, vomiting	Open surgery	5 yr	Bowel repair	Alive at 6 months	–	Mechanical factor
12	2008 Bognar et al. [22]	67 M	Rectal bleeding	Open surgery	4 yr	Aortic reconstruction, bowel repair	Alive at 24 days	+	Mechanical factor
13	2007 Broutzos et al. [32]	85 M	Gastrointestinal bleeding	Open surgery	10 yr	EVAR	Alive at 1 year	–	Unclear
14	2006 Heidemann et al. [33]	52 M	Hematochezia, hematemesis	Open surgery	6 m	Aortic reconstruction, bowel repair	Alive at 8 months	–	Unclear
15	2007 Tsunekawa et al. [23]	75 M	Fever, malaise	Open surgery	15 yr	Aortic reconstruction, bowel repair	Alive at 1 month	+	Infection
16	2006 Matemini et al. [14]	73 M	Melena	Open surgery	15 yr	EVAR	Alive	–	Unclear
17	2005 Mundal et al. [24]	82 F	Hematemesis	Open surgery	17 yr	Surgery	Died	+	Unclear
18	2004 French et al. [13]	68 F	Hematemesis	EVAR	1 yr	Aortic reconstruction, bowel repair, debridement	Died	–	Infection
19	2002 Tomlinson et al. [34]	90 M	Melena, abdominal pain	Open surgery	5 yr	EVAR	Alive at 14 months	–	Unclear
20	2000 Makar et al. [35]	70 M	Epigastric discomfort	EVAR	4 m	Antibiotics	Died	–	Unclear
21	1999 Karacagil et al. [36]	70 F	Melena, fever	Open surgery	14 yr	Aortic reconstruction, bowel repair	Alive at 2 years	–	Unclear
22	1993 Neergaard et al. [37]	69 M	Hematemesis, melena	Open surgery	8 yr	Aortic reconstruction, bowel repair	Alive at 4 months	–	Unclear
23	2018 Jiang et al. [38]	85 M	Melena, tiredness, dizziness, fever	EVAR	2 yr 4 m	Aortic reconstruction, bowel repair, debridement	Alive at 15 months	–	Unclear

Table 1 Summary of published case reports describing secondary aortoenteric fistula after both open surgery and endovascular aortic reconstruction for abdominal aortic aneurysm (*Continued*)

Date	Authors and reference number	Age, sex	Clinical presentation	Initial treatment for aneurysm	Period†	Treatment for SAEF	Outcome	Pathological examination‡	Estimated aetiology§
24	2017 Hansen et al. [12]	75 F	Melena	Open surgery	2 yr	Open surgery + EVAR	Alive at 12 years	–	Infection
25	1998 Yabu et al. [25]	77 M	Dyspnea	Open surgery	10 yr	None*	Died	+	Mechanical factor

EVAR endovascular aortic reconstruction, F female, M male, SAEF secondary aortoenteric fistula, yr year

†period after abdominal aortic aneurysm treatment

‡whether pathological examination was performed or not

§possible cause suggested by authors of the report

*patient died immediately before any treatment

intestinal wall in the usual fashion during surgery may only be effective up to a certain point, and that further adjuvant maneuvering may be necessary to maintain postoperative isolation. Adhesive barriers could be applied to cover the closing line of the retroperitoneum.

Clinically, SAEFs present in a variety of ways, which makes a prompt diagnosis challenging [3, 8]. Whereas gastrointestinal bleeding occurs in most such cases, sepsis occurs in only approximately half on initial presentation [5, 8, 27], which is possibly because such symptoms are unlikely if the fistula was formed mainly due to mechanical stimuli. Infection might occur later, however, through contact with intestinal bacteria. Therefore, an SAEF should be suspected in cases of bloody stool or hematemesis after AAA reconstruction, even in the absence of septic symptoms. If an SAEF is the suspected cause of bloody stool, immediate exploratory surgery should be considered to prevent potentially catastrophic developments, even if the general status of the patient is stable.

Conclusions

This case report described gastrointestinal bleeding due to an SAEF. Physical stimuli were associated with its formation due to adhesion between the aorta and the intestinal wall after AAA reconstruction, indicating the importance of keeping a reconstructed aorta isolated from the intestine. Clinicians should suspect SAEF in patients with bloody stool after aneurysm surgery.

Abbreviations

AAA: Abdominal aortic aneurysm; CT: Computed tomography; SAEF: Secondary aortoenteric fistula

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Availability of data and materials

The datasets generated during the current report are not publicly available due to privacy of information, but are available from the corresponding author on reasonable request.

Authors' contributions

Study conception: HS, YN, and MT. Writing: HS and YN. Critical review and revisions: all authors. Final approval of the article: all authors. Accountability for all aspects of the work: all authors.

Ethics approval and consent to participate

Not applicable.

Consent for publication

Written informed consent was obtained from the patient's next-of-kin for publication of this case report and any accompanying images. A copy of the written consent is available for review by the Editor-in-Chief of this journal.

Competing interests

The authors declare that they have no competing interests.

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